# Vitamin D Toxicity and Clinical Consequences of Hypervitaminosis

**Yuriy Fedorov** (Doctor of Medical Sciences, Professor, Vice-Rector for Scientific Work, Lviv Medical University, Ukraine)
**Alona Kovpak** (PhD in Pharmaceutical Sciences, Associate Professor, Deputy Vice-Rector for Scientific Work, Lviv Medical University, Ukraine)
Corresponding author: Alona Kovpak

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**Abstract.** Vitamin D is an essential fat-soluble vitamin that enters the human body with certain animal products, fortified foods, dietary supplements, and is also synthesized endogenously under the influence of solar radiation. It plays a critical role in regulating calcium-phosphorus metabolism and maintaining bone health, preventing the development of rickets and osteopenia. In recent years, there has been a growing body of evidence regarding its involvement in numerous metabolic processes and potential link to the risk of autoimmune diseases, cancer, cardiovascular disease, depression, dementia and infectious diseases.
However, despite the widespread use and availability of vitamin D, including in the form of over-the-counter supplements, there is a risk of developing hypervitaminosis D, which, although rare, can have serious clinical consequences. Vitamin D toxicity is usually caused by excessive intake of high doses of the vitamin (intentional or accidental), prescription errors, or lack of proper monitoring of vitamin D levels during treatment of certain diseases (e.g., osteoporosis, renal osteodystrophy, malabsorption).
Clinical manifestations of hypervitaminosis D are caused by hypercalcemia and can range from nonspecific symptoms (weakness, fatigue, anorexia, bone pain) to more serious neurological (confusion, apathy, ataxia), gastrointestinal (nausea, vomiting, constipation, pancreatitis), renal (polyuria, polydipsia, nephrolithiasis) and cardiac (arrhythmias) disorders. Treatment is mainly supportive and aimed at lowering calcium levels by discontinuing vitamin D and calcium intake, rehydration, and in severe cases, calcitonin, bisphosphonates, sometimes glucocorticoids, or hemodialysis.

**Keywords:** vitamin D, toxicity, hypervitaminosis, overdose, hypercalcemia, regulation of bone metabolism, side effects.

**Introduction.** Vitamin D is a unique fat-soluble vitamin that plays a fundamental role in maintaining calcium and phosphorus homeostasis, which is critical for bone health throughout the life of a person. It is obtained from a limited number of animal products (fatty fish, liver, egg yolks), fortified foods (dairy products, cereals), and is also synthesized endogenously in the skin under the influence of ultraviolet B radiation. The classical function of vitamin D is to ensure bone mineralization, and its deficiency leads to rickets in children and osteomalacia and osteoporosis in adults [1, 2].

The literature describes the role of ergocalciferol (vitamin D) in maintaining human health, of pharmacotherapy of covid, post-covid and long-covid disorders, in particular in the treatment of tuberculosis. Vitamin D performs several important functions in the body, including supporting bone health, the immune system and the cardiovascular system. The analysis shows that its addition to the complex therapy of tuberculosis can improve the recovery of patients and their quality of life. A review of the market for specialty food products with vitamin D was conducted, including various dosage forms, manufacturers, manufacturing countries and brands. The results indicate the preference for capsules and tablets, and also identify the main manufacturing countries, such as the USA, Poland and Germany [3-11].

Separate research was conducted on medical errors in the diagnosis and pharmacotherapy of health disorders [12].

However, in recent decades, scientific research has significantly expanded the understanding of the biological functions of vitamin D, revealing its numerous pleiotropic effects. Vitamin D receptors (VDRs) and enzymes involved in its metabolism are present in many tissues and organs not directly related to the bone system. This suggests that vitamin D is involved in the regulation of the immune response, cell proliferation and differentiation, inflammation, cardiovascular function and the nervous system. There is growing evidence of a potential link between vitamin D levels and the

risk of developing autoimmune diseases, certain types of cancer, tuberculosis cardiovascular disease, diabetes mellitus, infectious diseases, depression and dementia [2].

Amid growing awareness of the importance of vitamin D and the prevalence of vitamin D deficiency in the population, there has been a significant increase in the consumption of vitamin D supplements, which are widely available both with and without a prescription. This trend, while aimed at improving health, carries a potential risk of developing the opposite condition, hypervitaminosis D and intoxication. Vitamin D toxicity, although considered relatively rare, can occur as a result of intentional or accidental ingestion of excessively high doses, prescribing errors, or lack of proper laboratory monitoring in patients requiring high doses to treat certain conditions (e.g., malabsorption syndromes, after bariatric surgery, and certain forms of osteoporosis) [13, 14].

The pathophysiological basis of vitamin D toxicity is dysregulation of calcium metabolism, which leads to the development of hypercalcemia. It is the excess calcium in the blood that causes most clinical manifestations and symptoms of intoxication, which can range from mild and nonspecific (fatigue, weakness, anorexia) to severe, life-threatening damage to the kidneys, cardiovascular and nervous systems [15, 16].

**The purpose of the study** was to review in detail the etiology, pathophysiological mechanisms, clinical manifestations, diagnostic approaches and current treatment strategies for vitamin D toxicity. It also aims to emphasize the importance of healthcare professionals' awareness of the risks of hypervitaminosis D, the need for careful monitoring when prescribing high doses of the vitamin, and the role of interprofessional interaction in preventing this potentially dangerous condition.

**Materials and methods.** This article is an analytical review based on the analysis of current scientific and clinical literature on vitamin D metabolism, its toxicity, clinical manifestations of hypervitaminosis D and approaches to its treatment. The search for relevant sources was carried out in major scientometric databases, such as PubMed/MEDLINE, Google Scholar, Cochrane Library, using the following keywords: ‘vitamin D’, “toxicity”, “hypervitaminosis D”, “hypercalcemia”, “vitamin D overdose”, “treatment of vitamin D toxicity”, “vitamin D toxicity”, “hypervitaminosis D”, “vitamin D overdose”.

The analysis included review articles, clinical trials, meta-analyses, case reports, as well as current clinical guidelines and recommendations from leading medical societies (in particular, the Endocrine Society), published mainly in the last 10-15 years. The selected information was systematized according to the main aspects of the problem: etiology, pathophysiology, clinical picture, diagnosis, differential diagnosis, treatment, prognosis, complications and prevention of vitamin D toxicity. The main focus was on summarizing current data and forming a holistic view of the problem of hypervitaminosis D in clinical practice.

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**Results and discussion.** A review of the literature shows that vitamin D toxicity, although a relatively rare condition, most often occurs as a result of iatrogenic causes, such as excessive intake of high doses of vitamin D supplements (intentional or accidental), medication errors, or lack of proper monitoring of calcium and vitamin D levels in patients receiving high doses for the treatment of specific conditions (e.g., absorption disorders, osteoporosis, renal osteodystrophy). Much less commonly, it is caused by excessive consumption of fortified foods or endogenous hyperproduction of active vitamin D metabolites in granulomatous diseases or lymphomas. It is important to note that excessive sun exposure does not lead to vitamin D toxicity due to physiological mechanisms of regulation of its synthesis and inactivation in the skin.

Pathophysiologically, hypervitaminosis D is realized through the accumulation of high concentrations of vitamin D metabolites, in particular 25(OH)D, which exceeds the binding capacity of vitamin D-binding protein (VDBP). This leads to an increase in the free fraction of 1,25(OH)2D (calcitriol) and enhanced biological effects: increased intestinal calcium absorption, increased renal

calcium reabsorption and increased bone resorption. The end result of these processes is the development of hypercalcemia, which underlies most clinical manifestations of intoxication.

The clinical picture of vitamin D toxicity is caused by hypercalcemia and can be polymorphic and nonspecific, especially in the early stages. Generalized weakness, fatigue, anorexia, nausea, vomiting, constipation, and bone and muscle pain are common. With more severe hypercalcemia, neurological symptoms (confusion, apathy, agitation, less often stupor, coma), renal manifestations (polyuria, polydipsia, dehydration, nephrocalcinosis, nephrolithiasis, acute kidney injury), gastrointestinal complications (peptic ulcers, pancreatitis) and cardiac disorders (arrhythmias, shortening of the QT interval on the ECG) may develop.

Diagnosis of hypervitaminosis D requires a comprehensive approach. A thorough history of vitamin D and calcium intake (including over-the-counter supplements) is key. Laboratory testing usually reveals elevated serum total and/or ionized calcium levels (>11 mg/dL), suppressed parathyroid hormone (PTH) levels due to a negative feedback mechanism, and significantly elevated 25(OH)D levels (often >150 ng/ml). The level of 1,25(OH)2D may be normal or elevated. It is important to assess renal function and electrolyte levels. An ECG may reveal characteristic changes associated with hypercalcemia. Differential diagnostics is performed with other causes of hypercalcemia, such as primary hyperparathyroidism, malignant neoplasms, granulomatous diseases, thiazide diuretics, immobilization, etc.

Treatment of vitamin D toxicity is mainly supportive and aimed at correcting hypercalcemia and dehydration. The primary measures are immediate discontinuation of all vitamin D and calcium preparations, as well as ensuring adequate hydration (preferably with isotonic sodium chloride solution) to increase renal calcium excretion. Excessive restriction of physical activity should be avoided. In cases of severe symptomatic hypercalcemia (>14 mg/dl) or in the presence of complications, more active therapy may be required, including calcitonin (for a rapid but short-term effect) and intravenous bisphosphonates (pamidronate, zoledronic acid) for a longer-term reduction in bone resorption. The use of glucocorticoids (hydrocortisone, prednisone) is controversial, but can be effective in hypercalcemia associated with granulomatous diseases or lymphomas, as they reduce calcitriol levels. In cases of severe renal impairment or refractory hypercalcemia, hemodialysis may be required. It is mandatory to review the patient's medication prescriptions to adjust the dose of vitamin D in the future.

The prognosis of vitamin D toxicity is usually favorable with timely detection and adequate treatment, and most cases resolve without serious long-term consequences. However, severe hypercalcemia can lead to acute kidney damage, which sometimes requires temporary hemodialysis. Permanent kidney damage is a rare complication.

The discussion of these data highlights the importance of a balanced approach to vitamin D prescription and intake. The growing popularity of supplements and the prevalence of self-medication increase the risk of overdose. Healthcare professionals at all levels - doctors, pharmacists, nurses - play a key role in the prevention of hypervitaminosis D. This includes being aware of recommended doses, taking a thorough history of all medications and supplements, prescribing adequate doses according to clinical indications, performing appropriate monitoring (calcium, phosphorus, 25(OH)D levels) in patients on high doses, and educating patients about the potential risks of excessive vitamin D intake. Clear interprofessional communication and coordination is needed to avoid medication errors and ensure patient safety. Further research could be aimed at clarifying safe upper limits of vitamin D intake for different populations and developing clearer algorithms for monitoring therapy.

*Etiology*

In healthy individuals, vitamin D toxicity can result from taking excessive amounts of vitamin D, either intentionally or accidentally [15]. Prescription errors without frequent monitoring of vitamin D levels can also lead to toxicity [16]. Toxicity caused by lack of monitoring is often seen in patients requiring high doses for treatment of conditions such as osteoporosis, renal osteodystrophy, psoriasis, gastric bypass, celiac disease, or inflammatory bowel disease [14, 17].

Vitamin D is found in fish, meat, and dairy products, and the dose is rarely sufficient to cause toxicity [18]. Patients taking high doses of vitamin D and inadvertently consuming high amounts of fortified milk are also at increased risk of vitamin D toxicity. This may also result from excessive production of 1,25(OH)2D in medical conditions such as granulomatous diseases and lymphomas [19]. Excessive sun exposure does not cause vitamin D toxicity due to the regulation and conversion of vitamin D into its inactive metabolites [18]. Although mainly reported in animals, exposure to rodenticides containing cholecalciferol can also lead to vitamin D toxicity [20, 21].

*Pathophysiology*

Vitamin D is a fat-soluble vitamin that is stored in the liver and adipose tissue. It is available from exogenous sources and is synthesized endogenously by the human body. The Endocrine Society Clinical Practice Guidelines state that the daily requirement for vitamin D for adults aged 19 to 50 years is 600 IU/day. For people aged 50 to 70 years, the daily requirement is at least 600 IU/day. For people over 70 years, the daily requirement is at least 800 IU/day. The maximum recommended daily requirement is 4000 IU/day for anyone over 80 years [22].

In hypervitaminosis D, the concentrations of vitamin D metabolites, such as 25(OH)D; 24,25(OH)2D; 25,26(OH)2D; and 25(OH)D-26,23-lactone, are significantly increased [23]. Abnormally high concentrations of vitamin D metabolites exceed the binding capacity of VDBP, resulting in the release of free 1,25(OH)2D. It is believed that vitamin D toxicity is associated with elevated concentrations of 25(OH)D and free 1,25(OH)2D, although this hypothesis has yet to be conclusively proven [23].

*History and Physiology*

A detailed history is essential in making a diagnosis of vitamin D toxicity. A careful review of medication history, including use of over-the-counter supplements, is necessary.

Clinical signs and symptoms of vitamin D toxicity are due to the effects of hypercalcemia. Symptoms can often be nonspecific and subtle, such as weakness, fatigue, anorexia, and bone pain [24, 25]. More serious symptoms include neurological symptoms such as confusion, apathy, agitation, irritability, and occasionally ataxia, stupor, and coma. Gastrointestinal symptoms include abdominal pain, nausea, vomiting, constipation, peptic ulceration, and pancreatitis (from malignant calcifications). Renal symptoms include polyuria, polydipsia, and nephrolithiasis [26]. Severe hypercalcemia can also lead to cardiac arrhythmias.

Physical examination of patients with signs of intoxication may show loss of skin turgor and dryness of mucous membranes (due to dehydration); changes in mental status; and abdominal tenderness without rebound, rigidity, or guarding.

*Evaluation*

The diagnosis of vitamin D toxicity is based on a comprehensive assessment of the patient's medical history and clinical symptoms. Laboratory evaluation includes monitoring of serum calcium (often >11 mg/dL), ionized calcium, phosphate levels, and parathyroid hormone (PTH), which are often suppressed through a negative feedback loop [27].

In cases of vitamin D toxicity, serum 25(OH)D levels often exceed 150 ng/mL (375 nmol/L), accompanied by normal or elevated 1,25(OH)2D levels [14]. Patients with concomitant granulomatous diseases may have low or normal 25(OH)D levels and elevated 1,25(OH)2D levels. A basic metabolic panel should be performed to assess renal function for the potential effects of hypercalcemia and to assess any electrolyte imbalance caused by excessive vomiting.

The most common ECG finding is a shortened QT interval. In addition, the EGG may reveal a prolonged PR interval, a shortened ST segment, flattened T waves, and Osborn waves (J waves), as evidenced by a positive J-point deviation in the precardiac leads [28].

*Treatment*

Clinical treatment for vitamin D toxicity is primarily supportive and aimed at lowering calcium levels.

Vitamin D and calcium supplements should be discontinued. Excessive bed rest should also be avoided to prevent hypercalcemia due to immobilization.

✓ Isotonic saline should be used to correct dehydration and increase renal calcium clearance [24].

✓ In severe toxicity leading to severe hypercalcemia (serum calcium >14 g/dL), calcitonin and bisphosphonates may be used. The recommended dosage is as follows:

✓ Calcitonin at a dose of 4 U/kg intramuscularly can be administered and repeated every 12 hours for up to 48 hours.

✓ Concomitant intravenous bisphosphonates may be administered: pamidronate 90 mg intravenously over 2 hours and zoledronic acid 4 mg intravenously over 15 minutes [29, 30].

✓ Calcitonin and bisphosphonates shown to enhance the efficacy of calcitonin [31].

The use of intravenous glucocorticoids is controversial and is usually reserved for the treatment of vitamin D toxicity associated with granulomatous disease. This drug acts by reducing calcitriol levels, which in turn lowers plasma calcium levels. By reducing intestinal calcium absorption and increasing urinary calcium excretion, it helps regulate body calcium levels [32].

✓ Hydrocortisone 100 mg/day or prednisone 40 mg/day for 5 days.

✓ Patients may require hemodialysis for renal failure or correction of refractory hypercalcemia.

✓ The patient's medication list should be reviewed to adjust future doses of vitamin D supplements.

✓ Patients should be counseled to avoid excessive use of vitamin supplements.

*Differential diagnosis*

Vitamin D hypervitaminosis should be differentiated from other causes of hypercalcemia. Other diseases that mimic the signs and symptoms of hypervitaminosis:

- ✓ Malignant hypercalcemia
- ✓ Hypercalcemia in granulomatous diseases
- ✓ Primary, secondary and tertiary hyperparathyroidism
- ✓ Vitamin A toxicity
- ✓ Thyrotoxicosis
- ✓ Paget's disease
- ✓ Hypercalcemia caused by prolonged immobilization
- ✓ Milk-alkali syndrome

*Prognosis*

Most cases of vitamin D toxicity resolve without serious complications or sequelae. However, in some cases, severe hypercalcemia can lead to acute renal failure requiring hemodialysis. Permanent kidney damage from vitamin D toxicity is rare.

*Complications*

Complications of vitamin D toxicity rarely led to kidney failure requiring hemodialysis [33].

*Patient education and restraint*

Patient awareness of the harmful effects of excessive vitamin D supplementation is essential. To ensure effective treatment of underlying diseases, it is essential to educate patients, especially those taking high doses of vitamin D, about the importance of adherence to the prescribed regimen. In addition, patients should be informed of the importance of follow-up care while taking high-dose vitamin D supplements to reduce the risk of vitamin D toxicity. In certain situations, laboratory tests to assess calcium and vitamin D levels may be useful for monitoring.

*Improving healthcare team outcomes*

Preventing vitamin D toxicity requires a collaborative effort by an interprofessional healthcare team, including physicians, experienced practitioners, nurses, and pharmacists. Because vitamin D is commonly prescribed and sold over the counter, prescribers must be mindful of the different high-dose formulations and recommended daily requirements to prevent medication errors and adverse outcomes.

**Conclusions**. Vitamin D toxicity, although relatively rare, is a clinically significant condition that occurs mainly as a result of excessive intake of exogenous vitamin D, often due to prescription errors or uncontrolled use of dietary supplements. The main pathophysiological consequence of hypervitaminosis D is the development of hypercalcemia, which causes a wide range of clinical manifestations, from nonspecific symptoms to severe renal, cardiovascular and nervous system damage. Timely diagnosis based on anamnesis, clinical picture and laboratory data (elevated calcium and 25(OH)D levels, suppressed PTH) and adequate treatment aimed at correcting hypercalcemia and rehydration allow for a complete recovery in most cases. Prevention of vitamin D toxicity requires a balanced approach to supplementation, proper monitoring of patients at risk, and raising awareness of the potential dangers of excessive vitamin D intake among both healthcare professionals and the public. Effective interprofessional collaboration is needed to ensure the safe and rational use of vitamin D supplements.

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